

ANSWER 3 OF 4 MEDLINE on STN

1999424509. PubMed ID: 10496190. Racial differences in response to therapy for heart failure: analysis of the vasodilator-heart failure trials. Vasodilator-Heart Failure Trial Study Group. [Carson P; Ziesche S; Johnson G; Cohn J N. (Veteran's Affairs Medical Center, Washington, District of Columbia, USA.)] Journal of cardiac failure, (1999 Sep) 5 (3) 178-87. Journal code: 9442138. ISSN: 1071-9164. Pub. country: United States. Language: English.

AB BACKGROUND: Heart failure in blacks has been associated with a poorer prognosis than in whites. In such diseases as hypertension, blacks show pathophysiological differences and respond differently to some therapies than whites. The aim of this study is to evaluate the clinical characteristics and response to therapy of **black** compared with white patients with heart failure. METHODS AND RESULTS: In the first Vasodilator-Heart Failure Trial (V-HeFT I), 180 **black** male patients were compared with 450 white male patients for baseline characteristics, prognosis, and response to therapy. In V-HeFT II, the same comparisons were made for 215 **black** and 574 white male patients, including an analysis stratified by the presence or absence of a history of hypertension. In both trials, **black** patients had a lower incidence of coronary artery disease, greater incidence of previous hypertension, and a greater cardiothoracic ratio ($P < .05$) than white patients. In V-HeFT II, plasma norepinephrine levels were significantly less in blacks; plasma renin activity was less only in blacks with a history of hypertension. Overall mortality or hospitalization for congestive heart failure did not differ between blacks and whites in the placebo group in V-HeFT I. However, the mortality of **black** patients receiving hydralazine plus **isosorbide dinitrate** (H-I) was reduced ($P = .04$) in V-HeFT I, whereas white patients showed no difference from placebo. In V-HeFT II, only white patients showed a mortality reduction from enalapril therapy compared with H-I therapy ($P = .02$). Whites also showed evidence of greater blood pressure reduction and enhanced regression of cardiac size in response to enalapril. When stratified by history of hypertension in V-HeFT II, only whites with a history of hypertension, who had greater renin levels, showed significant mortality reduction with enalapril compared with H-I therapy. Hospitalization rates did not differ between treatment groups in either study. CONCLUSION: Whites and blacks showed differences in cause, neurohormonal stimulation, and pharmacological response in heart failure. This retrospective analysis suggests angiotensin-converting enzyme inhibitors are particularly effective in whites, and the H-I combination can be equally effective in blacks. Prospective trials involving large numbers of **black** patients are needed to further clarify their response to therapy.

L3 ANSWER 4 OF 4 MEDLINE on STN

83093394. PubMed ID: 6401366. Acute coronary vasospasm secondary to industrial nitroglycerin withdrawal. A case presentation and review. Przybojewski J Z; Heyns M H. South African medical journal. Suid-Afrikaanse tydskrif vir geneeskunde, (1983 Jan 29) 63 (5) 158-65. Journal code: 0404520. ISSN: 0038-2469. Pub. country: South Africa. Language: English.

AB A **Black** employee exposed to industrial nitroglycerin (NG) in an explosives factory presented with severe precordial pain. The clinical presentation was that of significant transient anteroseptal and anterolateral transmural myocardial ischaemia which responded promptly to sublingual **isosorbide dinitrate**. Despite being removed from exposure to industrial NG and receiving therapy with long-acting oral nitrates and calcium antagonists, the patient continued to experience repeated attacks of severe retrosternal pain, although transient myocardial ischaemia was not demonstrated electrocardiographically during these episodes. Cardiac catheterization revealed a normal myocardial haemodynamic system and selective coronary arteriography delineated coronary arteries free from any obstructive

lesions. An ergonovine (ergometrine) maleate provocative test failed to elicit coronary artery spasm, although this was undertaken while the patient was on nitrate and calcium-blocker therapy. Clinical records of previous significant constrictive pericarditis (probably due to tuberculosis) with resultant abnormalities on the ECG complicated the diagnosis. Evaluation was further hindered by the known "variant pattern" seen on the ECGs of members of the **Black** population. We postulate that this patient's clinical features were a direct result of severe vasospasm affecting the left coronary artery; it is also strongly suggested that withdrawal from contact with industrial NG precipitated this potentially lethal coronary vasospasm. The role played by industrial NG in ischaemic heart disease is reviewed, as well as the importance of the "normal variant pattern" in the assessment of cardiac disease in **Black** patients. As far as we are aware this is the first time that the use of the ergonovine maleate provocative test has been documented in the industrial NG withdrawal syndrome.

=>

00/24814
MA/

25 ANSWER 10 OF 28 BIOSIS COPYRIGHT 2000 BIOSIS

1994:440281 Document No.: PREV199497453281. Nitrates in congestive heart failure. Dupuis, Jocelyn. Montreal Heart Inst., 500 Belanger St. E., Montreal, PQ H1T 1C8 Canada. Cardiovascular Drugs

and Therapy, (1994) Vol. 8, No. 3, pp. 501-507. ISSN: 0920-3206. Language: English.

AB Nitrates are commonly used in the therapy of congestive heart failure (CHF). They exert beneficial hemodynamic effects by decreasing left ventricular filling pressure and systemic vascular resistance while modestly improving cardiac output. The improvement in left ventricular function caused by nitrates is the result of combined reduction in outflow resistance and mitral regurgitation, while decreased pericardial constraint and subendocardial ischemia may also contribute to the process. With continuous nitrate administration, complete arterial tolerance develops, while venous tolerance appears to

be only partial. The major mechanism of tolerance is loss of vascular smooth muscle sensitivity to nitrates. An increase in total blood volume occurring during the first few hours of an acute administration may

partly contribute to tolerance. The importance of reflex neurohumoral activation is controversial; although it may contribute to tolerance in CHF, its

role does not appear to be major. Chronic continuous nitrate therapy in CHF improves submaximal and maximal exercise tolerance. In combination therapy with hydralazine, isosorbide dinitrate reduces mortality, although to a lesser extent than the angiotensin converting enzyme inhibitor enalapril. Intravenous

or sublingual nitrates are first-line agents in the therapy of acute pulmonary edema. In severe CHF, refractory to standard medical therapy, a short course of intravenous nitroglycerin, with or without inotropic agents, can help break the vicious spiral of CHF. Because tolerance

occurs without nitrate-free intervals and until an optimal schedule of administration is determined, it makes good sense to include a nightly nitrate-free interval when prescribing nitrates for CHF in order to maintain maximal benefit during the hours of activity.